Neonatal viral diarrhoeas

IW CAPLE

University of Melbourne, Veterinary Clinical Centre, Princes Highway, Werribee, Victoria 3030

Introduction

Diarrhoea continues to be one of the more common and important causes of economic loss in young animals (Anon 1978). Virus particles identified as rotaviruses, coronaviruses, calici-like viruses, astroviruses, parvoviruses, and several others have been detected by direct electron microscopy of ultracentrifuged samples of diarrhoeic faeces from young animals and human infants over the past 20 years. Despite numerous studies on the many aetiological agents associated with neonatal viral diarrhoea in recent years (Tzipori 1985), the challenge for today's new veterinary graduate is similar to that faced by Bill Snowdon when he graduated from the University of Sydney in 1948. When investigating outbreaks of diarrhoea in calves, piglets, foals, kids, and lambs in the field, or in various species of wildlife kept in zoos, it is still difficult to know which bacteria, viruses or protozoa are responsible for the diarrhoea (Blood and Radostits 1989).

Rotaviruses

The reovirus-like virus (rotavirus) was first isolated from calves with diarrhoea in the United States twenty years ago (Mebius *et al* 1969). In 1973, the virus was associated with diarrhoea in calves (Turner *et al* 1973), and in human infants in Victoria (Bishop *et al* 1973). Subsequent studies showed rotavirus to be present in pigs (Rodger *et al* 1976), deer (Tzipori *et al* 1977), foals, lambs, kids, dogs, mice and rabbits (Tzipori 1985; Ellis and Daniels 1988). Experimental transmission in gnotobiotic neonates and detailed pathological studies have confirmed their pathogenic potential. The virus infects the villous epithelial cells of the small intestine and results in villous atrophy, loss of mucosal β -galactosidase, and an inability to digest lactose in milk (Halpin and Caple 1976).

Early studies on rotaviruses were hampered by the use of specific but rather insensitive electronmicroscopic techniques for detection of virus particles from ultracentrifuged diarrhoeic faeces. Enzyme-immunoassay tests (ELISA) provide a more sensitive means for detecting rotaviruses in faecal specimens (Ellis and Daniels 1988).

Most field strains of rotavirus were non-cultivable until it was shown that incorporation of trypsin into cell culture medium promoted viral replication. Now much is known about the viruses morphology, genome, capsid proteins, antigenic determinants, viral replication and genetics and biology (Holmes 1983; Tzipori 1985). On the basis of cross-reactivity of group antigen, genome profile analysis on polyacrylamide gel electrophoresis, and one-dimensional terminal fingerprinting of the RNA genome the rotaviruses detected to date have been classified into one of five groups: A, B, C, D and E. Group A rotaviruses is the major cause of diarrhoea in both human and animals. Other rotaviruses which are morphologically similar but antigenically distinct are referred to as atypical rotaviruses. One has been shown to be important in causing diarrhoea in adult people (Tao 1988). Another group C rotavirus has wide distribution in pigs (Chasey and Davies 1984; Fu 1987). Rotaviruses are generally considered to be host specific, but experimentally it has been shown that inter-species infection can occur.

Among the rotavirus strains there are different serotypes and subgroups (Tzipori 1985). This makes diagnosis and the preparation of suitable vaccines difficult. Repeated exposure of animals to different rotaviruses results in acquisition of

antibodies specific to each serotype which the animal has been previously exposed and does not broaden to include serotypes to which the animal has no previous experience. In beef herds it may be important to expose unmated heifers to possible new serotypes of rotaviruses introduced onto a farm by grazing them on the paddocks where calves have diarrhoea.

Diarrhoea in Calves

Rotaviruses vary in virulence in calves of the same age, and some are capable of causing diarrhoea in calves up to 3 months of age. The severity of rotavirus infection in young animals is influenced by the presence of colostral antibody in the intestine, and absorbed antibody is not protective. The morbidity rate in beef and dairy herds varies from herd to herd and from year to year and ranges from 5 to 80%. Mortality varies from 5% to 60% and probably depends on the level of colostral immunity in the calves, the prevalence of enteric colibacillosis and cryptosporidiosis, and the level of animal husbandry and clinical management provided in the herd. A study of outbreaks of diarrhoea on 9 dairy farms in Gippsland in 1981 showed that rotavirus was present in 6% of normal calves and in 49% of calves with diarrhoea (Jerret 1985). *Cryptosporidia* spp was present in 36% of the diarrhoeic calves, *Salmonella* spp was present in 12% and K99 *E. coli* present in 5%. More than one pathogen was present in 19% of the affected calves.

The importance of rotavirus in diarrhoea in calves is often questioned as the virus can be excreted by healthy calves and adult cows for several weeks. Coronavirus, which is an important cause of diarrhoea in calves in some countries, has been observed in Australia but is thought to be of low virulence (Tzipori 1985).

Protection against rotaviral disease is dependent on specific rotaviral antibody in the lumen of the intestine. Colostral serum antibody does not protect animals against clinical disease. One practical approach to the control of rotaviral diarrhoea in dairy calves is to include 1 per cent colostrum in milk or milk replacer fed during the first month.

Broad protection by colostrum is a reflection of repeated infections with different serotypes of rotavirus, and this broad response can be maintained by infection with a single serotype. Rotavirus vaccines developed overseas against diarrhoea in calves have included a modified live virus vaccine for oral administration to calves immediately after birth, and others administered intramuscularly to pregnant cows to enhance maternal lactogenic immunity to provide passive protection to the calf. Some have included both rotavirus and coronavirus strains, and others rotavirus and enterotoxigenic *E. coli* (Snodgrass *et al* 1988).

Diarrhoea in Piglets

Escherichia coli infection is the most important cause of diarrhoea in neonatal piglets in Australia (Fahy *et al* 1987). There are 2 distinct clinical syndromes: neonatal colibacillosis caused by non-haemolytic *E. coli* in piglets within the first 2 d of life, and post-weaning diarrhoea caused by haemolytic *E. coli* 4 to 7 d after weaning. Rotavirus infection which is widespread in the pig population, is occasionally associated with diarrhoea in piglets over 7 d of age, which have been weaned early and are devoid of maternal protection through

immunoglobulins in colostrum and milk (Rodger *et al* 1975; Tzipori and Williams 1978).

Other enteropathogens causing neonatal diarrhoea in piglets aged from 4 d to weaning include enteropathogenic *E. coli*, and coccidia. Transmissible gastroenteritis virus (TGE), and coronavirus (Porcine epidemic diarrhoea) have not been demonstrated in pigs with diarrhoea in Australia.

Diarrhoea in Foals

Rotavirus has been isolated from up to 40% of foals with diarrhoea between the ages of 5 d to 7 wk (Tzipori and Walker 1978). There are at least 2 distinct serotypes of equine rotavirus, and field strains may induce varying degrees of clinical illness in foals (Tzipori 1985). Other agents involved in diarrhoea in neonatal foals in Australia include *Streptococcus durans*, *Clostridium perfringens* type C, *Salmonella* spp, and possibly *Aeromonas* spp (Tzipori 1985).

Discussion

The approach to treatment and prevention of diarrhoea in young animals has changed little over the past 20 years. Most intestinal viral infections are self-limiting provided fluid and electrolyte balance of the young animal is not severely compromised, and other enteric pathogens such as *Cryptosporidia*, *Escherichia coli*, *Salmonella* spp, and *Campylobacter* spp are not present. Today's clinician relies on supportive therapy for affected neonates by administration of fluids, electrolytes and glucose to combat dehydration and energy deficits, systemic and oral antimicrobials to combat primary and secondary infections, and pays attention to environmental hygiene and temperature. Giving colostrum or hyperimmune serum orally provides passive protection until the animals develop a natural immunity against the pathogens involved.

Usually outbreaks of diarrhoea have resolved by the time the diagnostic laboratory has reported back its findings on pathogens detected or isolated from submitted faecal and blood samples. Most inputs into the study of viruses associated

with neonatal diarrhoea in animals and humans over the past decade has been in the laboratory identification of agents, and in the definition of agent and host interactions (Tzipori 1985). One major difficulty for the clinician and the diagnostic laboratory continues to be the availability of suitable diagnostic tests. Diagnosis requires detection of the virus of viral antigen, and/or the demonstration of a serological response. It is envisaged that advances from current research on the molecular biology of rotaviruses and other enteric pathogens may lead to improvements in diagnosis and our understanding of the epidemiology of neonatal viral diarrhoea. The eternal hope is that this research may also lead to the development of improved vaccines.

References

- Anon (1978) — *J Am Vet Med Assoc* 173: 515
Bishop RF, Davidson GP, Holmes IH and Ruck BJ (1973) — *Lancet* 2: 1281
Blood DC and Radostits OM (1989) — *Veterinary Medicine*, 7th edn, Balliere Tindall, London
Chasey D and Davies P (1984) — *Vet Rec* 114: 16
Ellis GR and Daniels E (1988) — *Aust Vet J* 65: 133
Fahy VA, Connaughton ID, Driesen SJ and Spicer EM (1987) — In *Pig Production*, Proc No 95, Post-Grad Ctee Vet Sci, Univ Sydney, p 965
Fu ZF (1987) — *NZ vet J* 35: 115
Halpin CG and Caple IW (1976) — *Aust Vet J* 52: 438
Holmes I (1983) — In *The Reoviridae*, Plenum, New York, p 359
Jerret IV (1985) — In *Dairy Cattle Production*, Proc No 78, Post-Grad Ctee Vet Sci, Univ Sydney, p 156
Mebus CA, Underdahl, NR, Rhodes MB and Twiehaus MJ (1969) — *Univ Nebr Afric Exp Stn Res Bull* 233: 1
Rodger SM, Craven JA and Williams IH (1975) — *Aust Vet J* 51: 536
Snodgrass DR, Angus KW, Herring AJ, Blewett DA, Scott FM and Cuthbertson JC (1988) — Annual Report, Moredun Research Institute, Scotland
Tao H (1988) — *Adv Virus Res* 35: 193
Tzipori S (1985) — *Adv Vet Sci Comp Med* 29: 103
Tzipori S, Caple IW and Butler R (1977) — *Vet Rec* 99: 398
Tzipori S and Walker M (1978) — *Aust J Exp Biol Med Sci* 56: 453
Tzipori S and Williams IH (1978) — *Aust Vet J* 54: 188
Turner AJ, Caple IW, Craven JA and Reingarnum C (1973) — *Aust Vet J* 49: 544